

CORRESPONDENCE

Re: Risk of Thyroid Cancer After Exposure to ^{131}I in Childhood

In his editorial, Boice (1) summarized “what is new” about radiogenic thyroid cancer in children as a result of the April 1986 Chernobyl nuclear accident, as presented by Cardis et al. (2). One of their puzzling findings is the fact that potassium iodide given to children months to years after exposure reduced the cancer risk by threefold. Cardis et al. hypothesize that the reduction in risk might be the result of shrinkage in the size of the thyroid due to prolonged administering of dietary iodine supplements.

Neither Cardis et al. nor Boice considered the fact that the release of ^{131}I (half-life = 8 days) into the environment from the molten fuel rods was accompanied by large amounts of ^{129}I (half-life = 16 million years), probably in higher proportion than that released by the Hanford, WA, plutonium production process (3).

In a report that suggests excess cancers, including thyroid cancers, among a population of residents downwind of the reactors in Hanford, WA, during periods of large releases of radioiodine into the atmosphere (downwinders), Grossman et al. (4) referred to several studies that show that the commonly assumed biological clearance half-time for iodine (80 days from the thyroid and 12 days for the rest of the body) (3) is inconsistent with several well-documented observations of much longer iodine retention times in tissue. Therefore, in contrast to statements that contributions to dose from uptake of ^{129}I by the thyroids of residents downwind of Hanford, WA, were negligible (3), we suggested that a long retention time, combined with the considerably higher relative biological effectiveness of the very low-energy radiation from the radioactive decay of ^{129}I compared with ^{131}I emissions may contribute substantially to damage of thyroid tissue, including cancer induction (4).

The sizable reduction of risk by administering potassium iodide to chil-

dren long after exposure to Chernobyl fallout supports our hypothesis of an important role of ^{129}I in thyroid cancer induction, mitigated by long-term metabolic exchange between stable and radioactive iodine, and would explain the puzzle presented by the findings of Cardis et al.

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RESPONSES

We do not share the opinion expressed by Grossman and Nussbaum that “... the release of ^{131}I (half-life = 8 days) into the environment from the molten fuel rods was accompanied by large amounts of ^{129}I (half-life = 16 million years), probably in higher proportion than that released by the Hanford, WA, plutonium production process.” The ratio of activities of ^{129}I to ^{131}I in the Unit 4 reactor core at the time of the accident on 26 April 1986 has been estimated (via different approaches that were based on the fuel burned or on measurements in soil) to be almost eight orders of magnitude

lower than that of ^{131}I , i.e., in the range of 1.3×10^{-8} to 2.6×10^{-8} (1–3).

We have used the standard dose coefficients recommended of the International Commission on Radiological Protection in publications 67 and 71 (4,5), which are based on the biological clearance half-time values in these publications, to calculate the expected ratio of thyroid dose from intake of ^{129}I to thyroid dose from intake of ^{131}I for the 2-month period after the accident (the period over which virtually all of the dose from ^{131}I was accumulated). This ratio is on the order of 10^{-6} for adults and 10^{-7} for children. Thus, the average thyroid dose from intake of ^{129}I in subjects in our study can be estimated to be on the order of 0.1 μGy ; this dose is very small in comparison with the average dose from ^{131}I , which is on the order of 400 mGy for children younger than 2 years at the time of the accident and 40 mGy for children 10–14 years old at the time of the accident (6).

In the years since the accident, intake of ^{129}I from Chernobyl fallout continues to occur with ingestion of locally produced foodstuffs (mainly with milk). The deposition density of ^{129}I from the Chernobyl accident measured in the most contaminated regions in Belarus ranged from 80 mBq/m² to 2.8 kBq/m² in evacuated settlements of the 30-km zone around the Chernobyl nuclear power plant (3). If we apply a radioecological model describing transfer of radioactivity from soil through plants and milk to humans (7), we obtain ^{129}I thyroid doses from the intake of milk of up to 1 $\mu\text{Gy}/\text{year}$ in the most contaminated regions. Thus, even over a 50-year period, the estimated cumulative doses from ^{129}I would be considerably lower than that from ^{131}I . Even with much longer iodine retention times in tissue, as mentioned by Grossman and Nussbaum, we expect that doses from ^{129}I intake would be considerably smaller than doses from intake of ^{131}I .

We do not, therefore, share their opinion that ^{129}I plays an important role in the large increase in the incidence of thyroid cancer in young people after the Chernobyl accident.

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The contribution of ^{129}I to thyroid dose compared with that of ^{131}I released during the Chernobyl accident in 1986 and with the background radiation from natural sources is minor because of the long physical half-life (15.7 million

years) of ^{129}I and its short retention time in the body (1). There is no experimental evidence for a carcinogenic effect of ^{129}I —the rate of decay is one billion times lower than that of ^{131}I and is much too low to expect one. Exposure of ^{129}I to rats at 1 cGy/day for life produced no excess of thyroid tumors or other thyroidal effects (2). ^{129}I occurs naturally when high-energy particles interact with xenon in the upper atmosphere, and ^{129}I is produced after nuclear detonations and in nuclear reactors. ^{129}I is not considered a principal radioactive release product after Chernobyl (3). Any continued small exposure to ^{129}I to those alive in 1986 would be at older ages than occurred for the short-lived radioiodines, and any carcinogenic risk would accordingly be reduced because risk decreases dramatically with age at exposure. The possible contribution of ^{129}I to thyroid dose from the Hanford nuclear site releases in Washington State was estimated to be very small (4), and comprehensive epidemiologic studies of populations residing near Hanford have failed to identify an increased risk of thyroid cancer (or any of 14 measures of thyroid disease) associated with ^{131}I exposure (5).

The areas near Chernobyl of highest fallout to radioactive iodines were also the areas most deficient in dietary stable iodine. The continued administration of potassium iodine (KI) reduced thyroid cancer risk, even after the time when blockage of ^{131}I uptake was no longer possible. How this risk reduction could occur is not entirely clear. It has been postulated that diets deficient in iodine may interact with radioactive iodines to enhance the risk of thyroid cancer (6). Animal studies have shown that the tumorigenic effect of thyroid irradiation depends on the duration and extent of subsequent thyroid stimulation; i.e., thyroid stimulation resulting from iodine-deficient diets increased the number of radioiodine-induced thyroid tumors, and radiation-induced tumors were conversely reduced when excessive thyroid stimulation was removed (7). It is conceivable that restoring normal levels of stable iodine to the diet in areas of endemic goiter might quell the overactive thyroid gland so that any underlying damage from prior radioiodine exposures did not progress to cancer. If KI administration has influenced the patterns of thyroid cancer risk among children living near Chernobyl, this influence is unlikely

because of any protective action against a hypothetical and implausible carcinogenic effect of ^{129}I . It is more plausible that the higher levels of thyroxine associated with iodine supplements decreased the level of thyroid stimulation (and subsequent cancer risk) by indirect inhibition of thyrotropin secretion.

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